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(FILE 'HOME' ENTERED AT 19:02:30 ON 18 JUN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 19:04:13 ON 18 JUN 2003

L1 0 S ZUCJER(3A)FATTY(4A)RAT
L2 2067 S ZUCKER(3A)FATTY(4A)RAT
L3 557739 S DIABETES OR RESTENOSIS
L4 532 S L2(S)L3
L5 36788 S HUMAN(5A)(DIABETES OR RESTENOSIS OR ATHEROSCLEROSIS)
L6 24 S L4 AND L5
L7 12 DUP REM L6 (12 DUPLICATES REMOVED)

=> d bib ab 1-12 l7

L7 ANSWER 1 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2003:86429 BIOSIS
DN PREV200300086429
TI Intramyocellular lipid and insulin resistance: A longitudinal in vivo 1H-spectroscopic study in Zucker Diabetic Fatty rats.
AU Kuhlmann, Johanna; Neumann-Haefelin, Claudia; Belz, Ulrich; Kalisch, Juergen; Juretschke, Hans-Paul; Stein, Marion; Kleinschmidt, Elke; Kramer, Werner; Herling, Andreas W. (1)
CS (1) Disease Group Metabolic Diseases, Aventis Pharma Deutschland GmbH, Industriepark Hoechst, Pharmacology H 815/H 821, 65926, Frankfurt/Main, Germany: andreas.herling@aventis.com Germany
SO Diabetes, (January 2003, 2003) Vol. 52, No. 1, pp. 138-144. print. ISSN: 0012-1797.
DT Article
LA English
AB Insulin resistance plays an important role in the pathogenesis of **human** type 2 **diabetes**. In **humans**, a negative correlation between insulin sensitivity and intramyocellular lipid (IMCL) content has been shown; thus, IMCL becomes a marker for insulin resistance. Recently, magnetic resonance spectroscopy (MRS) has been established as a dependable method for selective detection and quantification of IMCL in humans. To validate the interrelation between insulin sensitivity and IMCL in an animal model of type 2 **diabetes**, we established volume selective 1H-MRS at 7 Tesla to noninvasively assess IMCL in the **rat**. In male obese **Zucker** Diabetic **Fatty rats** and their lean littermates, IMCL levels were determined repeatedly over 4 months, and insulin sensitivity was measured by the euglycemic-hyperinsulinemic clamp method at 6-7 and at 22-24 weeks of age. A distinct relation between IMCL and insulin sensitivity was demonstrated as well as age dependence for both parameters. Rosiglitazone treatment caused a clear reduction of IMCL and hepatic fat despite increased body weight, and a marked improvement of insulin sensitivity. Thus, the insulin sensitizing properties of rosiglitazone were consistent with a redistribution of lipids from nonadipocytic (skeletal muscle, liver) back into fat tissue.

L7 ANSWER 2 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
AN 2002:468173 BIOSIS
DN PREV200200468173
TI In vivo phosphorylation of insulin receptor substrate 1 at serine 789 by a novel serine kinase in insulin-resistant rodents.
AU Qiao, Li-ya; Zhande, Rachel; Jetton, Thomas L.; Zhou, Gaochao; Sun, Xiao Jian (1)
CS (1) Dept. of Medicine, University of Vermont, Given Bldg., C-350, Burlington, VT, 05405: xsun@zoo.uvm.edu USA
SO Journal of Biological Chemistry, (July 19, 2002) Vol. 277, No. 29, pp.

26530-26539. http://www.jbc.org/. print.
ISSN: 0021-9258.
DT Article
LA English
AB Insulin resistance is a key pathophysiologic feature of obesity and type 2 **diabetes** and is associated with other **human** diseases, including **atherosclerosis**, hypertension, hyperlipidemia, and polycystic ovarian disease. Yet, the specific cellular defects that cause insulin resistance are not precisely known. Insulin receptor substrate (IRS) proteins are important signaling molecules that mediate insulin action in insulin-sensitive cells. Recently, serine phosphorylation of IRS proteins has been implicated in attenuating insulin signaling and is thought to be a potential mechanism for insulin resistance. However, in vivo increased serine phosphorylation of IRS proteins in insulin-resistant animal models has not been reported before. In the present study, we have confirmed previous findings in both JCR:LA-cp and **Zucker fatty rats**, two genetically unrelated insulin-resistant rodent models, that an enhanced serine kinase activity in liver is associated with insulin resistance. The enhanced serine kinase specifically phosphorylates the conserved Ser789 residue in IRS-1, which is in a sequence motif separate from the ones for MAPK, c-Jun N-terminal kinase, glycogen-synthase kinase 3 (GSK-3), Akt, phosphatidylinositol 3'-kinase, or casein kinase. It is similar to the phosphorylation motif for AMP-activated protein kinase, but the serine kinase in the insulin-resistant animals was shown not to be an AMP-activated protein kinase, suggesting a potential novel serine kinase. Using a specific antibody against Ser(P)789 peptide of IRS-1, we then demonstrated for the first time a striking increase of Ser789-phosphorylated IRS-1 in livers of insulin-resistant rodent models, indicating enhanced serine kinase activity in vivo. Taken together, these data strongly suggest that unknown serine kinase activity and Ser789 phosphorylation of IRS-1 may play an important role in attenuating insulin signaling in insulin-resistant animal models.

L7 ANSWER 3 OF 12 MEDLINE DUPLICATE 3
AN 2002251589 MEDLINE
DN 21986614 PubMed ID: 11991215
TI Normal perivascular sensory dilator nerve function in arteries of Zucker diabetic fatty rats.
AU Pamarthi Mohan F; Rudd M Audrey; Bukoski Richard D
CS Cardiovascular Disease Research Program, Julius L. Chambers Biomedical/Biotechnology Research Institute, North Carolina Central University, Durham 27707, USA.
NC HL59868 (NHLBI)
HL64761 (NHLBI)
SO AMERICAN JOURNAL OF HYPERTENSION, (2002 Apr) 15 (4 Pt 1) 310-5.
Journal code: 8803676. ISSN: 0895-7061.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200210
ED Entered STN: 20020507
Last Updated on STN: 20021026
Entered Medline: 20021024
AB BACKGROUND: Type II **diabetes** in **humans** is associated with pathology of both the cardiovascular and peripheral sensory nervous systems. Because abnormal vasodilator responses have been reported in animals of type II **diabetes** and perivascular sensory nerves are a source of vasodilator substances, we tested the hypothesis that sensory nerve-dependent relaxation is abnormal in arteries of the **Zucker** diabetic **fatty** (ZDF) **rat** model of type II **diabetes**. METHODS: The ZDF rats and genetic controls were studied at 26 weeks of age. Tail-cuff systolic blood pressure (BP) was measured,

serum was obtained for chemical determinations, and mesenteric branch arteries were isolated for wire myograph analysis and confocal-based measurement of calcitonin gene-related peptide (CGRP) positive nerve density. RESULTS: No differences in BP were detected. Serum glucose, triglycerides, and cholesterol were significantly elevated in ZDF. Sensory nerve-dependent vasodilation was assessed by measuring relaxation of phenylephrine preconstricted arterial segments to cumulative addition of divalent calcium ion (Ca2+) or capsaicin. Neither Ca(2+)-nor capsaicin-induced relaxation were different in ZDF versus control (maximal ZDF response to Ca2+ = 64% +/- 2% v 59% +/- 4%; ED50 for Ca2+ = 3.7 +/- 0.5 mmol/L v 3.2 +/- 0.5 mmol/L; n = 5, P = not significant [NS]; maximal ZDF response to capsaicin = 68% +/- 9% v 74% +/- 4%; ZDF ED50 = 3.8 +/- 0.5 nmol/L v 9.8 +/- 7 nmol/L; n = 5, P = NS). In contrast, the maximal relaxation response to acetylcholine was impaired in ZDF (maximal ZDF response = 83% +/- 5% v 94% +/- 2%, n = 4, P = .039; ED50 for acetylcholine = 8.1 +/- 2.9 nmol/L for ZDF v 33.5 +/- 18.2; n = 4 per group, P = .086). The CGRP positive nerve density was not different between groups. CONCLUSIONS: Blood pressure, perivascular sensory nerve CGRP content, and dilator function is normal in the ZDF model of type II diabetes, whereas endothelium-dependent relaxation is impaired.

L7 ANSWER 4 OF 12 MEDLINE DUPLICATE 4
AN 2001156938 MEDLINE
DN 21100022 PubMed ID: 11156947
TI Diseases of liporegulation: new perspective on obesity and related disorders.
AU Unger R H; Orci L
CS Gifford Laboratories, Touchstone Center for Diabetes Research, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, Texas 75390-8854, USA.. runger@mednet.swmed.edu
NC DK02700-37 (NIDDK)
SO FASEB JOURNAL, (2001 Feb) 15 (2) 312-21. Ref: 90
Journal code: 8804484. ISSN: 0892-6638.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010322
AB Obesity-related diseases now threaten to reach epidemic proportions in the United States. Here we review in a rodent model of genetic obesity, the fa/fa **Zucker** diabetic **fatty** (ZDF) **rat**, the mechanisms involved in the most common complications of diet-induced **human** obesity, i.e., noninsulin-dependent **diabetes** mellitus, and myocardial dysfunction. In ZDF rats, hyperphagia leads to hyperinsulinemia, which up-regulates transcription factors that stimulate lipogenesis. This causes ectopic deposition of triacylglycerol in nonadipocytes, providing fatty acid (FA) substrate for damaging pathways of nonoxidative metabolism, such as ceramide synthesis. In beta cells and myocardium, the resulting functional impairment and apoptosis cause diabetes and cardiomyopathy. Interventions that lower ectopic lipid accumulation or block nonoxidative metabolism of FA and ceramide formation completely prevent these complications. Given the evidence for a similar etiology for the complications of human obesity, it would be appropriate to develop strategies to avert the predicted epidemic of lipotoxic disorders.

L7 ANSWER 5 OF 12 MEDLINE DUPLICATE 5
AN 2001092981 MEDLINE
DN 21023288 PubMed ID: 11147796

TI A genetic defect in beta-cell gene expression segregates independently from the fa locus in the ZDF rat.
AU Griffen S C; Wang J; German M S
CS Hormone Research Institute, Department of Medicine, University of California, San Francisco, USA.
NC DK02619 (NIDDK)
DK09377 (NIDDK)
DK48281 (NIDDK)
SO DIABETES, (2001 Jan) 50 (1) 63-8.
Journal code: 0372763. ISSN: 0012-1797.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200101
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010125
AB Type 2 diabetes is a strongly genetic disorder resulting from inadequate compensatory insulin secretion in the face of insulin resistance. The **Zucker** diabetic **fatty** (ZDF) **rat** is a model of type 2 **diabetes** and, like the **human** disease, has both insulin resistance (from a mutant leptin receptor causing obesity) and inadequate beta-cell compensation. To test for an independently inherited beta-cell defect, we examined beta-cell function in fetuses of ZDF-lean rats, which have wild-type leptin receptors. beta-Cell number and insulin content do not differ among wild-type, heterozygous, and homozygous ZDF-lean fetuses. However, insulin promoter activity is reduced 30-50% in homozygous ZDF-lean fetal islets, and insulin mRNA levels are similarly reduced by 45%. This is not a generalized defect in gene expression nor an altered transfection efficiency, because the islet amyloid polypeptide promoter and viral promoters are unaffected. Insulin promoter mapping studies suggest that the defect involves the critical A2-C1-E1 region. This study demonstrates that the ZDF rat carries a genetic defect in beta-cell transcription that is inherited independently from the leptin receptor mutation and insulin resistance. The genetic reduction in beta-cell gene transcription in homozygous animals likely contributes to the development of diabetes in the setting of insulin resistance.

L7 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1998:108583 CAPLUS
DN 128:226047
TI Troglitazone lowers islet fat and restores beta cell function of Zucker diabetic fatty rats
AU Shimabukuro, Michio; Zhou, Yang-Ting; Lee, Young; Unger, Roger H.
CS Gifford Lab. Cent. Diabetes Res., Dep. Internal Med., Univ. Texas Southwestern Med. Cent., Dallas, TX, 75235, USA
SO Journal of Biological Chemistry (1998), 273(6), 3547-3550
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB The thiazolidinedione compd. troglitazone, which is used to treat non-insulin-dependent **diabetes** mellitus (NIDDM) in **humans**, is also effective in the adipogenic NIDDM of **Zucker** diabetic **fatty** (ZDF) **rats**. To test the lipotoxicity hypothesis which attributes the pancreatic .beta.-cell dysfunction in adipogenic NIDDM to an excessive accumulation of fat in the pancreatic islets, we sought to det. if troglitazone-mediated amelioration of .beta.-cell function in islets of ZDF rats might be assocd. with a redn. in their elevated triglyceride (TG) content. Troglitazone (10 .mu.M) in the culture medium reduced the TG content of pancreatic islets from 7-wk-old male ZDF rats by 52%; this was reflected by decreased esterification and increased oxidn. of [3H]palmitate.

Glycerol-3-phosphate acyltransferase mRNA fell by 57% and acyl-CoA synthetase mRNA by 67% (brain isoform) and 38% (liver isoform), all consistent with the effects of troglitazone on TG metab. The 52% decrease in islet TG was accompanied by >30- and 2-fold improvements in glucose- and arginine-stimulated insulin secretion, resp. Thus, troglitazone exerts direct lipopenic activity in normal pancreatic islets and in islets of obese prediabetic ZDF rats; in the latter, this correlates with improvements in .beta.-cell function. The results are consistent with the lipotoxicity hypothesis for adipogenic diabetes.

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1998:561460 CAPLUS
DN 129:187522
TI Zucker diabetic fatty (ZDF) rats
AU Fujiwara, Toshihiko; Araki, Kazushi; Yorikane, Eiko; Hagisawa, Yuka; Fukushige, Junichiro; Horikoshi, Hiroyoshi
CS 1st Biol. Res. Lab., Sankyo Co., Ltd., Tokyo, 140, Japan
SO Diabetes Frontier (1998), 9(4), 455-458
CODEN: DIFREZ; ISSN: 0915-6593
PB Medikaru Rebyusha
DT Journal; General Review
LA Japanese
AB A review, with 8 refs, on pathophysiol. characteristics of ZDF rat having missense mutation of leptin receptor as a model of **human** type 2 **diabetes**. Hyperglycemia assocd. with hyperglycemia, diabetic complications, etc. are discussed.

L7 ANSWER 8 OF 12 MEDLINE DUPLICATE 6
AN 1999074421 MEDLINE
DN 99074421 PubMed ID: 9852229
TI The role of 12-lipoxygenase in pancreatic -cells (Review).
AU Bleich D; Chen S; Gu J L; Nadler J L
CS Division of Diabetes, Endocrinology and Metabolism, City of Hope National Medical Center, Duarte, CA 91010, USA.
SO INTERNATIONAL JOURNAL OF MOLECULAR MEDICINE, (1998 Jan) 1 (1) 265-72.
Ref: 72
Journal code: 9810955. ISSN: 1107-3756.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990316
AB Leukocyte type 12-lipoxygenase (12-LO) catalyzes the conversion of arachidonic acid (AA; C20:4) to 12-hydroperoxyeicosatetraenoic acid (12-HPETE) and linoleic acid (LA; C18:2) to 13-hydroperoxyoctadecadienoic acid (13-HPODE). Previous studies have demonstrated that 12-LO, but not 5- or 15-lipoxygenase (5-LO, 15-LO respectively), is specifically expressed in pancreatic -cells and is involved in regulating glucose-stimulated insulin secretion. Lipoxygenase products also have been linked with inflammatory pathways in endothelial cells, kidney mesangial cells, inflammatory bowel disease, and corneal epithelial cells. Therefore, 12-LO may play a role in cytokine mediated inflammation in pancreatic beta-cells (i.e. beta -cell dysfunction and cytotoxicity). Cytokines such as IL-1 stimulate both de novo 12-LO protein synthesis and enzyme activity in pancreatic beta-cells. The products generated by 12-LO may ultimately be involved in cellular events that lead to lipid peroxidation. Hydroperoxide and free radical production in beta-cells can

activate intracellular signaling pathways that lead to cell death or may directly damage mitochondrial and plasma membranes. Increased 12-LO expression has also been found in islets from prediabetic **Zucker fatty rats**, a model that demonstrates insulin secretory defects similar to **human** type 2 **diabetes**. In this review, we present an overview of the 12-LO pathway in regulating glucose-stimulated insulin secretion in beta-cells as well as more recent data which supports the hypothesis that the 12-LO pathway participates in cytokine mediated beta-cell dysfunction and cytotoxicity.

L7 ANSWER 9 OF 12 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 95:393847 SCISEARCH
GA The Genuine Article (R) Number: RB212
TI PROTEIN-KINASE-C IS INCREASED IN THE LIVER OF **HUMANS** AND RATS WITH NONINSULIN-DEPENDENT **DIABETES**-MELLITUS - AN ALTERATION NOT DUE TO HYPERGLYCEMIA
AU CONSIDINE R V (Reprint); NYCE M R; ALLEN L E; MORALES L M; TRIESTER S; SERRANO J; COLBERG J; LANZAJACOBY S; CARO J F
CS THOMAS JEFFERSON UNIV, JEFFERSON MED COLL, DEPT MED, DIV ENDOCRINOL & METAB, 1025 WALNUT ST, PHILADELPHIA, PA, 19107 (Reprint); THOMAS JEFFERSON UNIV, JEFFERSON MED COLL, DEPT SURG, PHILADELPHIA, PA, 19107
CYA USA
SO JOURNAL OF CLINICAL INVESTIGATION, (JUN 1995) Vol. 95, No. 6, pp. 2938-2944.
ISSN: 0021-9738.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 45
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We tested the hypothesis that liver protein kinase C (PKC) is increased in non-insulin-dependent **diabetes** mellitus (NIDDM). To this end we examined the distribution of PKC isozymes in liver biopsies from obese individuals with and without NIDDM and in lean controls, PKC isozymes alpha, beta, epsilon and zeta were detected by immunoblotting in both the cytosol and membrane fractions, Isozymes gamma and delta were not detected, There was a significant increase in immunodetectable PKC-alpha (twofold), -epsilon (threefold), and -zeta (twofold) in the membrane fraction isolated from obese subjects with NIDDM compared with the lean controls, In obese subjects without NIDDM, the amount of membrane PKC isozymes was not different from the other two groups. We next sought an animal model where this observation could be studied further. The **Zucker** diabetic **fatty rat** offered such a model system, Immunodetectable membrane PKC-alpha, -beta, -epsilon, and -zeta were significantly increased when compared with both the lean and obese controls, The increase in immunodetectable PKC protein correlated with a 40% elevation in the activity of PKC at the membrane, Normalization of circulating glucose in the rat model by either insulin or phlorizin treatment did not result in a reduction in membrane PKC isozyme protein or kinase activity, Further, phlorizin treatment did not improve insulin receptor autophosphorylation nor did the treatment lower liver diacylglycerol, We conclude that liver PKC is increased in NIDDM, a change that is not secondary to hyperglycemia. It is possible that PKC-mediated phosphorylation of some component in the insulin signaling cascade contributes to the insulin resistance observed in NIDDM.

L7 ANSWER 10 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1995:388584 BIOSIS
DN PREV199598402884
TI Endothelial-dependent vasodilation is preserved in non-insulin-dependent Zucker fatty diabetic rats.
AU Bohlen, H. Glenn (1); Lash, Julia M.
CS (1) Dep. Physiol. Biophysics, Indiana Univ. Med. Sch., 635 Barnhill Drive, Indianapolis, IN 46202 USA

SO American Journal of Physiology, (1995) Vol. 268, No. 6 PART 2, pp. H2366-H2374.
ISSN: 0002-9513.
DT Article
LA English
AB Alterations in the structural properties of the microvasculature and in vasodilation mediated by endothelial- and, to some extent, nonendothelial-dependent mechanisms occurs in insulin-dependent diabetic humans and animals. Less severe problems of this type appear to occur during non-insulin-dependent **diabetes** mellitus (NIDDM) in **humans**, but data based on animal models of NIDDM are not available. The endothelial- and nonendothelial-mediated dilation of intestinal arterioles was studied in insulin-resistant male **Zucker fatty** diabetic (DB) **rats** and their lean normal male littermates (LM) at ages 22-25 and 35-40 wk. DB become hyperglycemic (450-550 mg/100 ml) at age 9-10 wk. Microiontophoretic release of acetylcholine, ADP, and nitroprusside onto arterioles caused equivalent dilation in LM and DB for both large and intermediate diameter arterioles. Administration of streptozotocin (STZ) to DB at age 18-19 wk lowered their insulin concentration apprx 25% but did not significantly effect the resting plasma glucose concentration. However, endothelial-dependent vasodilation was attenuated by 70-80% within 8-10 wk. The overall results indicate that prolonged hyperglycemia in insulin-resistant but hyperinsulinemic rats does not impair the endothelial- and nonendothelial-dependent dilation of the intestinal microvasculature. However, compromising beta-cell function with STZ, as indicated by lowering the insulin concentration by one-fourth, substantially compromises endothelial-dependent dilation similar to that found in insulin-dependent diabetic rats and humans.

L7 ANSWER 11 OF 12 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 95:449033 SCISEARCH
GA The Genuine Article (R) Number: RE373
TI ENDOTHELIAL-DEPENDENT VASODILATION IS PRESERVED IN NON-INSULIN-DEPENDENT ZUCKER FATTY DIABETIC RATS
AU BOHLEN H G (Reprint); LASH J M
CS INDIANA UNIV, SCH MED, DEPT PHYSIOL & BIOPHYS, 635 BARNHILL DR, INDIANAPOLIS, IN, 46202 (Reprint).
CYA USA
SO AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, (JUN 1995) Vol. 37, No. 6, pp. H2366-H2374.
ISSN: 0363-6135.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 20
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Alterations in the structural properties of the microvasculature and in vasodilation mediated by endothelial- and, to some extent, nonendothelial-dependent mechanisms occurs in insulin-dependent diabetic humans and animals. Less severe problems of this type appear to occur during non-insulin-dependent **diabetes** mellitus (NIDDM) in **humans**, but data based on animal models of NIDDM are not available. The endothelial- and nonendothelial-mediated dilation of intestinal arterioles was studied in insulin-resistant male **Zucker fatty** diabetic (DB) **rats** and their lean normal male littermates (LM) at ages 22-25 and 35-40 wk. DB become hyperglycemic (450-550 mg/100 ml) at age 9-10 wk. Microiontophoretic release of acetylcholine, ADP, and nitroprusside onto arterioles caused equivalent dilation in LM and DB for both large and intermediate diameter arterioles. Administration of streptozotocin (STZ) to DB at age 18-19 wk lowered their insulin concentration similar to 25% but did not significantly effect the resting plasma glucose concentration. However, endothelial-dependent vasodilation was attenuated by 70-80% within 8-10 wk. The overall results

indicate that prolonged hyperglycemia in insulin-resistant but hyperinsulinemic rats does not impair the endothelial- and nonendothelial-dependent dilation of the intestinal microvasculature. However, compromising beta-cell function with STZ, as indicated by lowering the insulin concentration by one-fourth, substantially compromises endothelial-dependent dilation similar to that found in insulin-dependent diabetic rats and humans.

L7 ANSWER 12 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1995:430537 BIOSIS
DN PREV199598444837
TI Parathyroid hypertensive factor (PHF) and ionic changes in **Zucker fatty rats**: Parallels with **human** non-insulin dependent **diabetes**.
AU Lewanczuk, R. Z.
CS Univ. Alberta, Edmonton, AB Canada
SO Clinical and Investigative Medicine, (1995) Vol. 18, No. 4 SUPPL., pp. B74.
Meeting Info.: Annual Meeting of the Canadian Society for Clinical Investigation and the Royal College of Physicians and Surgeons of Canada Montreal, Quebec, Canada September 13-17, 1995
ISSN: 0147-958X.
DT Conference
LA English

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